The Impact of Basal Septal Hypertrophy on Outcomes after Transcatheter Aortic Valve Replacement

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Background: The role of basal septal hypertrophy (BSH) on preprocedural transthoracic echocardiography in transcatheter aortic valve replacement (TAVR) is unknown.

Methods: Medical charts and preprocedural transthoracic echocardiograms of 378 patients who underwent TAVR were examined. The association between BSH and the primary composite outcome of valve pop-out, recapture, embolization, aborted procedure, conversion to open procedure, new conduction disturbance, or need for permanent pacemaker ≤30 days after TAVR was evaluated. Patients with preexisting pacemakers were excluded. Sensitivity analyses were performed varying the definition of BSH.

Results: Of 296 TAVR patients (78.3%) with interpretable images, 55 (18.6%) had BSH at a median of 40 days (interquartile range, 19–62 days) before TAVR. Age and sex were similar among those with and without BSH. BSH patients received postdilation more frequently (BSH+ vs BSH−: 41.8% vs 29.9%, $P = .04$). A total of 50 individuals (16.9%) received pacemakers within 30 days, and 128 (43.2%) developed conduction disturbances (with left bundle branch block most common), without differences between groups. BSH was unrelated to the primary outcome on multivariate analysis (adjusted odds ratio BSH+ vs BSH−, 0.94; 95% CI, 0.42−2.11; $P = .88$).

Conclusions: In this convenience sample of TAVR recipients at a large academic medical center, patients with BSH were more likely to receive postdilation. BSH was not associated with procedural or conduction outcomes after TAVR in patients without preexisting pacemakers. (J Am Soc Echocardiogr 2019;32:1416-25.)

Keywords: Transcatheter aortic valve replacement, Echocardiography, Pacemaker

Within the past decade, transcatheter aortic valve replacement (TAVR) has emerged as an alternative to surgical aortic valve replacement for patients at intermediate to high surgical risk with symptomatic severe aortic stenosis.[1-4] With growth in the use of TAVR, increasing attention has been given to TAVR-related complications, particularly the development of complete heart block and the need for a permanent pacemaker (PPM).[5] Despite improvements in TAVR technology and increasing experience with implementation, the use of PPMs has not declined but in fact has increased.[6]

In this setting, it has been hypothesized that basal septal hypertrophy (BSH), a localized thickening of the basal portion of the left ventricular septum that associates with increased age and long-standing

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Abbreviations

BIDMC = Beth Israel Deaconess Medical Center

BSH = Basal septal hypertrophy

ESV = Edwards SAPIEN valve

MCV = Medtronic CoreValve

PPM = Permanent pacemaker

TAVR = Transcatheter aortic valve replacement

TTE = Transthoracic echocardiography

TVT = Transcatheter Valve Therapy

hypertension,[7-12] could contribute to the development of conduction and mechanical complications after TAVR.[11,13-16] The basal septum is in close proximity to both the aortic valve and the bundle of His, and localized hypertrophy at this site could contribute to procedural difficulty during TAVR and an increased risk for conduction disturbance and periprocedural complications, including PPM placement, valve pop-out, need for valve recapture, device embolization, conversion to an open procedure, or need to abort the procedure.[13-15] Despite the possible concerns about BSH's impact on outcomes, there exist few data on the outcomes of patients with BSH undergoing TAVR.

We therefore conducted a retrospective chart review of individuals undergoing TAVR at Beth Israel Deaconess Medical Center (BIDMC) to evaluate the impact of BSH on periprocedural and 30-day mechanical and electrical complications.

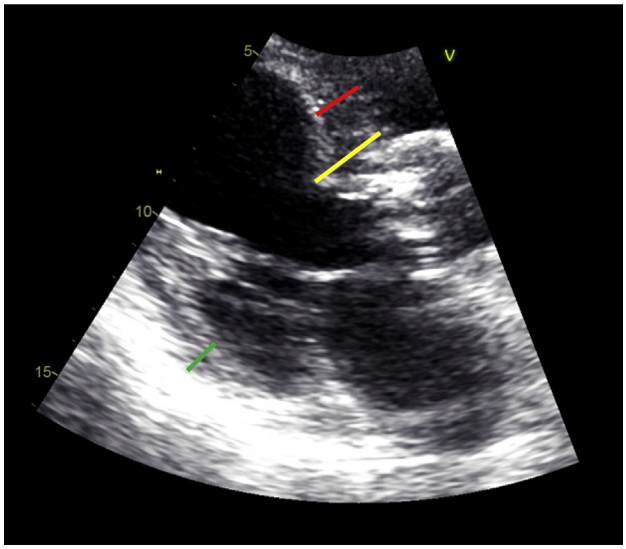

Figure 1 Schematic illustrating two-dimensional linear measurements done on preprocedural echocardiograms. *Yellow line* indicates left ventricular basal septal wall thickness, *red line* indicates left ventricular mid septal wall thickness, and *green line* indicates left ventricular posterior wall thickness. All measurements were obtained at end-diastole in the parasternal long-axis view.

METHODS

Study Population

We retrospectively evaluated adults (≥18 years old) who underwent TAVR at a single large academic medical center, BIDMC, from January 1, 2012, to December 31, 2016. BIDMC is a participating site in the Society of Thoracic Surgeons/American College of Cardiology Transcatheter Valve Therapy (TVT) Registry,[11] so patient demographics, comorbidities, and periprocedural information for all TAVR recipients are stored in a clinical database that is subsequently reviewed for accuracy by a full-time clinical nurse and uploaded to the TVT Registry website. This dataset was queried to identify TAVR participants who underwent transthoracic echocardiography (TTE) <3 months before their procedure dates. Those who did not undergo preprocedural TTE or for whom the echocardiographic images were deemed uninterpretable were excluded. The study was approved by the BIDMC institutional review board.

Covariates and Outcomes

Echocardiographic Variables. Preprocedural echocardiograms were manually reviewed by two physicians (N.J.K., G.C.S.) who received training in linear two-dimensional measurements by a board-certified echocardiographer (J.B.S.). These two physicians manually performed measurements of the left ventricular basal and mid interventricular septum and posterior wall on each subject. We defined BSH according to the definition used in the Framingham Heart Study as the presence of all four of the following criteria: (1) upper septal knuckle by visual assessment, (2) upper interventricular septal thickness ≥ 1.4 cm, (3) upper septal thickness/midseptal thickness ≥ 1.3, and (4) absence of wall motion abnormalities or scar in the midseptum that could result in isolated septal thickening (Figure 1).[11] All echocardiographic measurements were made using two-dimensional images, using a leading edge–to–trailing edge convention, in the parasternal long-axis view, at end-diastole, consistent with American Society of Echocardiography guidelines.[17] Images were acquired using GE E-95 or Vivid S70 machines (GE Medical Systems, Waukesha, WI), and measurements were made using GE EchoPAC version 201. All images deemed to be uninterpretable were reviewed a second time by the physician (N.J.K., G.C.S.) who did not review the initial study.

Additionally, several other echocardiographic variables were obtained from direct linkage to the BIDMC clinical echocardiographic database, which contains information on structural and functional variables included in echocardiographic reports from 2000 to 2018, all adjudicated by board-certified attending echocardiographers according to American Society of Echocardiography guidelines.[17] These variables included height, weight, left ventricular ejection fraction (using the biplane method of disks to define ventricular volumes), estimated right ventricular systolic pressure, left ventricular diastolic and systolic linear dimensions, left atrial volume index (using the biplane method of disks), number of aortic valve leaflets, etiology of aortic valve disease (degenerative, endocarditis, or other), presence and degree of aortic insufficiency, aortic valve peak velocity and gradient, aortic valve area, transaortic mean gradient, annular size, presence and degree of mitral regurgitation and stenosis, presence and degree of tricuspid regurgitation and stenosis, ascending aortic dimension, left ventricular outflow tract resting gradient, mitral valve peak E-wave and A-wave velocities and deceleration time by pulsed-wave Doppler at the mitral valve leaflet tips, and mitral valve septal and lateral annular tissue Doppler e′ measurements.

Clinical and Demographic Variables. Demographic, clinical, and comorbidity variables were determined at the date of procedure, using adjudicated TVT Registry variables, and included age, sex, race, insurance type, discharge disposition, body mass index, history of infective endocarditis, prior percutaneous coronary intervention or

HIGHLIGHTS

- Basal septal hypertrophy (BSH) is present in 18.6% of patients receiving TAVR.
- BSH has been postulated to associate with complications after TAVR.
- In this study, patients with BSH received post-dilation more frequently.
- In this study, BSH was not associated with adverse outcomes at 30 days post TAVR.

coronary artery bypass grafting, number of prior cardiac surgical procedures including prior aortic valve and nonaortic valve procedures, history of stroke or transient ischemic attack, carotid stenosis, peripheral arterial disease, recent smoking, hypertension, presence of and therapy for diabetes, home oxygen use, hostile chest, immunocompromised state, prior myocardial infarction, heart failure in the past 2 weeks and New York Heart Association functional class, presence of cardiogenic shock or cardiac arrest within 24 hours, porcelain aorta, atrial fibrillation or flutter, Society of Thoracic Surgeons surgical risk score,[18] indication for TAVR, results of a 5-min walk test, hemoglobin, creatinine, platelet count, international normalized ratio, albumin, bilirubin, forced expiratory volume in 1 sec and diffusing capacity for carbon monoxide, and receipt and results of diagnostic coronary angiography before TAVR. Additionally, missing TVT Registry variables and several additional variables were obtained from medical chart review by two physicians (N.J.K., G.C.S.) and included TAVR implantation for research or commercial use, brand and model, access site or approach, valve size, implantation depth (for both the noncoronary and left coronary cusps), presence of valve calcification (defined as binary presence or absence of valvular calcification on echocardiography), presence and type of preexisting arrhythmia, and contrast volume. TAVR brands and models included the balloon-expandable Edwards SAPIEN XT and SAPIEN 3 valves (ESV; Edwards Lifesciences, Irvine, CA), the self-expanding Medtronic CoreValve and Evolut-R (MCV; Medtronic, Minneapolis, MN), the Boston Scientific Lotus valve (Boston Scientific, Boston, MA), and the Direct Flow Medical device (Direct Flow Medical, Santa Rosa, CA), the latter of which is no longer in production.

Outcomes were determined according to definitions established by the Valve Academic Research Consortium–2. The primary outcome was a composite of valve pop-out, recapture, embolization, aborted procedure, conversion to an open procedure, new conduction disturbance, or need for PPM within 30 days of the procedure. Secondary outcomes included each individual outcome as well as postimplantation transaortic mean gradient, development of arrhythmia after the procedure, type of conduction disturbance after the procedure, and grade of paravalvular leak. All outcomes were determined from medical chart review by two physicians (N.J.K., G.C.S.). New conduction disturbance was determined through manual review of all electrocardiograms from 24 hours before TAVR to 24 hours following TAVR and again at 1-month follow-up by a board-certified cardiologist (G.M.B.).

Statistical Analysis

All patients with preexisting pacemakers were excluded from the analysis because of difficulty determining new conduction disturbance. Baseline characteristics of individuals with and without BSH are presented as mean ± SD for continuous variables and as frequencies and percentages for categorical variables and were compared using Student's *t* test for continuous variables and the χ^2 or Fisher exact test for categorical variables. The distribution of upper septal thickness by presence of BSH was plotted using a histogram, and quantiles were determined. Primary and secondary outcomes were compared between those with and without BSH using

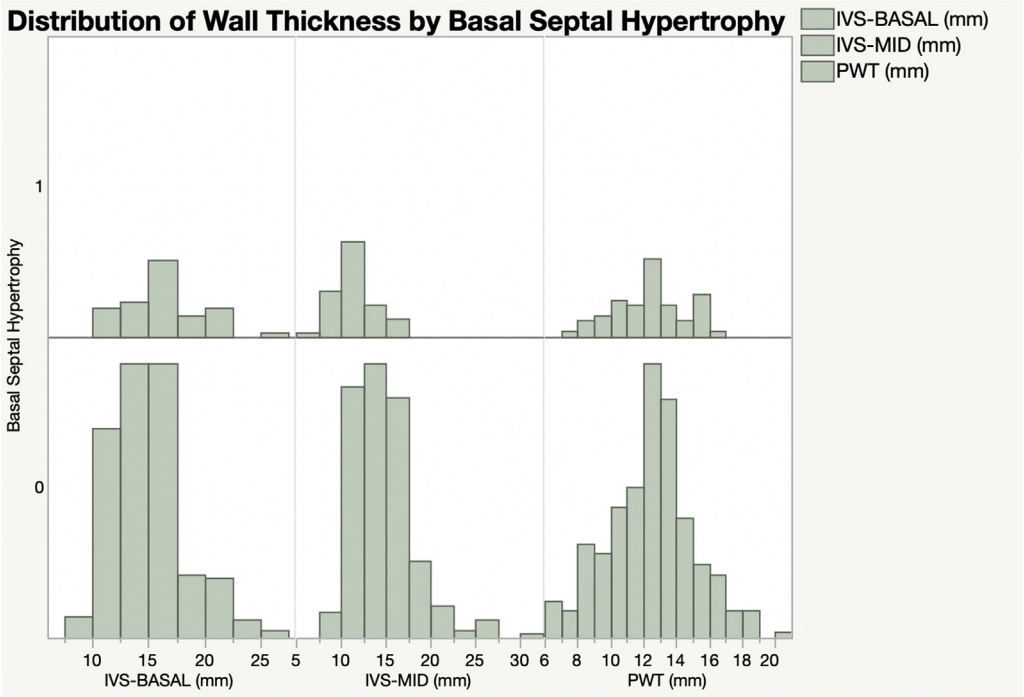

Figure 2 Histograms of upper septal, midseptal, and posterior wall thickness by presence of BSH.

Table 1 Characteristics of included patients

Variable	BSH+ (*n* = 55)	BSH− (*n* = 241)	*P*
Demographics			
Age, y	83.5 ± 10.2	82.8 ± 8.3	.66
Sex, female	28 (50.9)	117 (48.5)	.22
Race			.80
White	20 (36.4)	87 (36.1)	
Black	0 (0.0)	1 (0.4)	
Hispanic	0 (0.0)	1 (0.4)	
Insurance			.89
Medicare	15 (27.3)	68 (28.2)	
Private	5 (9.1)	22 (9.1)	
Medicaid	0 (0.0)	1 (0.4)	
Body mass index, kg/m^2	27.0 ± 5.0	27.8 ± 6.9	.31
Discharge disposition			.88
Extended care or rehabilitation center	11 (20.0)	68 (28.2)	
Home	19 (34.5)	94 (39.0)	
Hospice	0 (0.0)	1 (0.4)	
Skilled nursing facility	0 (0.0)	1 (0.4)	
Comorbidities			
Aortic valve disease etiology			.30
Degenerative	49 (89.0)	236 (97.9)	
Endocarditis	0 (0.0)	2 (0.8)	
Other	2 (3.6)	3 (1.2)	
Aortic valve morphology			.53
Bicuspid/ bicommissural	0 (0.0)	4 (1.7)	
Tricuspid/ tricommissural	38 (69.1)	203 (84.2)	
Uncertain	13 (23.6)	47 (19.5)	
Degree of aortic insufficiency			.40
None	7 (12.7)	40 (16.6)	
Trivial	8 (14.5)	56 (23.2)	
Mild	24 (43.6)	111 (46.1)	
Moderate	8 (14.5)	39 (16.2)	
Severe	4 (7.3)	7 (2.9)	
Degree of mitral insufficiency			.65
None	1 (1.8)	3 (1.2)	
Trivial	7 (12.7)	33 (13.7)	
Mild	17 (30.9)	114 (47.3)	
Moderate	19 (34.5)	73 (30.3)	
Severe	5 (9.1)	15 (6.2)	
History of mitral stenosis	6 (10.9)	18 (7.5)	.25
Degree of tricuspid regurgitation			.31
None	7 (12.7)	15 (6.2)	
Physiologic	11 (0.2)	61 (25.3)	
Mild	16 (29.1)	99 (41.1)	
Moderate	14 (25.5)	64 (26.6)	
Severe	2 (3.6)	15 (6.2)	
Prior PCI	12 (21.8)	82 (34.0)	.25
Prior CABG	6 (10.9)	62 (25.7)	.06
History of other cardiac procedure	7 (12.7)	25 (10.4)	.57
Number of prior cardiac surgical procedures	0.2 ± 0.5	0.3 ± 0.5	.65
History of myocardial infarction	8 (14.5)	41 (17.0)	>.99
History of heart failure in 2 wk before procedure	30 (54.5)	164 (68.0)	.53
NYHA functional class			.83
I	1 (1.8)	2 (0.4)	
II	10 (18.2)	42 (17.4)	
III	37 (67.3)	192 (79.7)	
IV	3 (5.5)	15 (6.2)	
Current dialysis	1 (1.8)	9 (3.7)	>.99
History of cerebrovascular event	7 (12.7)	35 (14.5)	>.99
History of transient ischemic attack	1 (1.8)	19 (7.9)	.22
Presence of carotid stenosis			
Bilateral carotids	3 (5.5)	26 (10.8)	.44
Left carotid	1 (1.8)	18 (7.5)	.22
Right carotid	4 (7.3)	19 (7.9)	>.99
None	15 (27.3)	44 (18.3)	.09
Not assessed	11 (20.0)	37 (15.4)	.30
History of peripheral arterial disease	7 (12.7)	40 (16.6)	.83
History of recent smoking	1 (1.8)	12 (5.0)	.70
History of hypertension	42 (76.4)	202 (83.8)	.77
History of diabetes	12 (21.8)	78 (32.4)	.40
Diabetes therapy			.35
Diet	2 (3.6)	9 (3.7)	
Insulin	3 (5.5)	23 (9.5)	
Oral	4 (7.3)	40 (16.6)	
None	12 (21.8)	44 (18.3)	
History of infective endocarditis	0 (0.0)	4 (1.7)	>.99
Chronic lung disease severity			.86

(*Continued*)

Table 1 (*Continued*)

Variable	BSH+ (*n* = 55)	BSH− (*n* = 241)	*P*
None	37 (67.3)	195 (80.9)	
Mild	7 (12.7)	32 (13.3)	
Moderate	3 (5.5)	9 (3.7)	
Severe	4 (7.3)	19 (7.9)	
Home oxygen use	4 (7.3)	19 (7.9)	>.99
Presence of a hostile chest	3 (5.5)	9 (3.7)	.43
Presence of a porcelain aorta	3 (5.5)	29 (12.0)	.32
Immunocompromised	6 (10.9)	48 (19.9)	.31
Cardiogenic shock within 24 h of procedure	0 (0.0)	2 (0.4)	>.99
Cardiac arrest within 24 h of procedure	0 (0.0)	0 (0.0)	>.99
History of preexisting pacemaker	5 (9.1)	48 (19.9)	.16
Preprocedure evaluation			
Surgical risk severity category			.97
Low risk	1 (1.8)	3 (1.2)	
Intermediate risk	1 (1.8)	6 (2.5)	
High risk	32 (58.2)	164 (68.0)	
Extreme risk	17 (30.9)	79 (32.8)	
Performance of 5MWT			.88
Not performed	17 (30.9)	85 (35.3)	
Unable to walk	4 (7.3)	20 (8.3)	
Yes	30 (54.5)	142 (58.9)	
5MWT result, min			
First attempt	9.5 ± 7.4	9.7 ± 6.4	.89
Second attempt	9.2 ± 7.1	9.6 ± 6.8	.74
Third attempt	9.1 ± 6.6	9.3 ± 4.4	.86
STS risk score, %	6.5 ± 3.6	7.1 ± 5.4	.38
Hemoglobin, g/dL	11.5 ± 1.8	11.1 ± 1.9	.18
Creatinine, mg/dL	1.1 ± 0.6	1.4 ± 0.9	.002
Platelets (no. per 100,000)	119.6 ± 123.0	128.0 ± 111.2	.65
International normalized ratio	1.2 ± 0.3	1.2 ± 0.3	.61
Albumin, g/dL	3.9 ± 0.6	3.9 ± 0.4	.82
Bilirubin, mg/dL	0.6 ± 0.5	0.6 ± 0.4	.99
FEV_1 (% predicted)	71.6 ± 24.6	71.5 ± 27.3	.99
DLCO (% predicted)	72.2 ± 23.6	76.6 ± 33.1	.60
Underwent diagnostic coronary angiography	45 (81.8)	241 (100.0)	.11
Number of diseased vessels on coronary angiography*			.20
0	24 (43.6)	88 (36.5)	
1	8 (14.5)	64 (26.6)	
2	8 (14.5)	40 (16.6)	
3	8 (14.5)	60 (25.0)	
LMCA stenosis*	4 (7.3)	28 (11.6)	.80
Proximal LAD stenosis*	3 (5.5)	45 (18.7)	.05

5MWT, 5-min walk test; *CABG*, coronary artery bypass graft; *DLCO*, diffusing capacity for carbon monoxide; *FEV_1*, forced expiratory volume in 1 sec, *LAD*, left anterior descending coronary artery; *LMCA*, left main coronary artery; *NYHA*, New York Heart Association; *PCI*, percutaneous coronary intervention; *STS*, Society for Thoracic Surgeons.

Data are expressed as mean ± SD or as number (percentage).

*Denominator and percentages reflect only those undergoing coronary angiography.

univariate statistics as above. Multivariate logistic regression was used to identify predictors of the primary composite outcome using age, body mass index, creatinine, hemoglobin, BSH, and left ventricular ejection fraction as predictors, chosen on the basis of their known or suspected associations with the primary outcome in the literature. Implantation depth was not included in the model, because of incomplete variable information. To isolate the effects of BSH on nonconduction outcomes, an additional multivariate logistic regression model was built to predict the composite outcome without conduction disturbance or pacemaker receipt using the same variable list. All analyses were done using JMP Pro version 13 and SAS version 9.4 (SAS Institute, Cary, NC) using a two-tailed P value $< .05$ to define statistical significance.

RESULTS

Overall Results

A total of 378 individuals underwent TAVR during the study period, of whom 296 (78.3%) had interpretable and nonmissing echocardiographic images. Echocardiograms were obtained at a median of 40 days (interquartile range, 19 to 62 days) before the date of TAVR. BSH was present in 55 patients (18.6%), with a mean upper septal thickness of 16.0 ± 3.3 mm compared with 14.6 ± 3.2 mm in those without BSH ($P = .006$; Figure 2). Baseline demographic and clinical data are described in Table 1. Age and sex were similar in patients with and those without BSH (BSH+ vs BSH−: mean age, 83.5 ± 10.2 vs 82.8 ± 8.3 years [$P = .66$]; female gender, 50.9% vs 48.5% [$P = .22$]). Patients with BSH had lower measured creatinine (BSH+ vs BSH−: mean creatinine, 1.1 ± 0.6 vs 1.4 ± 0.9 mg/dL; $P = .002$). All other characteristics, including baseline Society of Thoracic Surgeons surgical risk score, were similar between patients with and those without BSH.

Baseline Characteristics on Medical Chart Review and Preprocedural Echocardiography

Baseline clinical and procedural characteristics not available in the TVT Registry data were obtained via medical chart review

Table 2 Preprocedural and procedural characteristics obtained by medical chart review according to the presence of BSH

Variable	BSH+ (*n* = 55)	BSH− (*n* = 241)	*P*
History of preexisting arrhythmia or conduction system disease*	33 (0.6)	169 (70.1)	.65
Right bundle branch block	9 (16.4)	41 (17.0)	.66
Left anterior fascicular block	8 (14.5)	34 (14.1)	.72
Left bundle branch block	5 (9.1)	17 (7.1)	.85
Atrial fibrillation	11 (20.0)	68 (28.2)	.25
Atrial flutter	1 (1.8)	3 (1.2)	.69
Complete heart block	1 (1.8)	15 (6.2)	.21
First-degree atrioventricular block	8 (14.5)	39 (16.2)	.59
Second-degree atrioventricular block: Mobitz I	0 (0.0)	2 (0.8)	.75
Second-degree atrioventricular block: Mobitz II	1 (1.8)	2 (0.8)	.92
Intraventricular conduction delay	2 (3.6)	16 (6.6)	.38
Sick sinus syndrome	0 (0.0)	2 (0.8)	.69
Procedure done as part of research protocol	21 (38.2)	91 (37.8)	.64
TAVR brand			.64
Boston Scientific	0 (0.0)	6 (2.5)	
Direct Flow Medical	1 (1.8)	3 (1.2)	
Edwards Lifesciences	17 (30.9)	72 (29.9)	
Medtronic	37 (67.3)	185 (76.8)	
Arterial access approach			.67
Subclavian	0 (0.0)	3 (1.2)	
Transaortic	3 (5.5)	20 (8.3)	
Transapical	2 (3.6)	5 (2.1)	
Transfemoral	50 (90.9)	238 (98.8)	
Valve size, mm	27.4 ± 2.5	27.5 ± 2.8	.89
Implantation depth from noncoronary cusp of aortic valve	4.4 ± 1.8	4.2 ± 2.3	.48
Implantation depth from left coronary cusp of aortic valve	4.9 ± 1.7	4.8 ± 2.2	.62
Presence of annular calcification	36 (65.5)	177 (73.4)	.77
Performance of predilation	20 (36.4)	103 (42.7)	.76
Performance of postdilation	23 (41.8)	72 (29.9)	.04

Data are expressed as mean ± SD or as number (percentage).
*Individuals with multiple conduction system abnormalities or arrhythmias may be listed more than once.

(Table 2). The most commonly used device was the self-expanding MCV, followed by the balloon-expandable ESV. A transfemoral approach was used in the vast majority of TAVR recipients (BSH+ vs BSH−: 90.9% vs 98.8%, $P = .67$). Patients with BSH received postdilation more frequently than those without (BSH+ vs BSH−: 41.8% vs 29.9%, $P = .04$) but had similar implantation depths and proportions with annular calcification. On subgroup analysis, the finding of increased postdilation was present only in those receiving MCVs (BSH+ vs BSH−: MCV, 56.8% vs 33.9% [$P = .01$]; ESV, 11.8% vs 14.1% [$P = .80$]).

Echocardiographic characteristics of TAVR recipients are listed in Table 3. Patients with BSH had greater degrees of upper septal wall thickness but similar degrees of posterior wall thickness. Mitral valve peak E-wave velocity was higher in those with BSH (BSH+ vs BSH−: mean, 12.1 ± 3.6 vs 10.6 ± 3.3 cm/sec; $P = .01$). Other echocardiographic parameters were similar between those with and those without BSH.

Unadjusted Outcomes

Table 4 details the unadjusted periprocedural and 30-day mechanical and conduction outcomes by presence of BSH. The primary composite outcome occurred in 22 of those with BSH (40.0%) and 116 of those without BSH (48.1%; $P = .62$). There were no differences in rates of valve pop-out, recapture, aborted procedure, conversion to an open procedure, postimplantation mean gradient, or device embolization between those with and without BSH. New conduction disturbance occurred in 20 of those with BSH (36.4%) and 108 of those without BSH (44.8%; $P = .55$) with new left bundle branch block being most common (BSH+ vs BSH−: 25.5% vs 24.5%, $P = .76$), followed by complete heart block (BSH+ vs BSH−: 14.5% vs 13.7%, $P = .75$). Among those with interpretable echocardiograms in whom BSH could be measured, nine of those with BSH and 41 of those without BSH (16.4% vs 17.0%, $P = .41$) required PPMs <30 days after the procedure. There was no difference in degree of

Table 3 Echocardiographic variables on pre-TAVR echocardiography by presence or absence of BSH

Variable	BSH+ (*n* = 55)	BSH− (*n* = 241)	*P*
Height, in	165.6 ± 11.1	165.1 ± 11.6	.83
Weight, lb	75.7 ± 18.7	74.6 ± 17.3	.76
Annular dimension, cm	24.7 ± 2.3	25.1 ± 2.7	.56
Interventricular septal wall thickness at base level, mm	16.0 ± 3.3	14.6 ± 3.2	.006
Interventricular septal wall thickness at midlevel, mm	11.0 ± 2.2	14.3 ± 3.3	<.001
Posterior wall thickness, mm	11.8 ± 2.2	12.1 ± 4.4	.44
Left ventricular ejection fraction, %	54.4 ± 12.6	51.7 ± 15.3	.31
Left ventricular diastolic dimension, cm	4.7 ± 0.9	4.6 ± 0.8	.56
Left ventricular systolic dimension, cm	3.3 ± 1.1	3.1 ± 0.8	.24
Left atrial volume indexed, mL/m^2	38.3 ± 6.7	44.8 ± 9.4	.23
Estimated right ventricular systolic pressure by Doppler, mm Hg	54.5 ± 11.6	50.1 ± 15.2	.351
Aortic valve hemodynamics			
Peak velocity, m/sec	4.0 ± 0.8	4.1 ± 0.7	.51
Peak gradient, mm Hg	69.4 ± 22.5	70.3 ± 24.4	.85
Aortic valve area, cm^2	0.7 ± 0.2	0.7 ± 0.2	.26
Mean gradient, mm Hg	43.7 ± 14.8	43.6 ± 14.8	.98
Ascending aortic dimension, cm	3.2 ± 0.4	3.3 ± 0.5	.12
Mitral valve peak E velocity, cm/sec	12.1 ± 3.6	10.6 ± 3.3	.01
Mitral valve peak A velocity, cm/sec	9.9 ± 4.2	9.8 ± 4.0	.93
Mitral valve E-wave deceleration time, msec	203.4 ± 76.7	229.4 ± 89.5	.045
Septal mitral annular peak e′ velocity, cm/sec	5.6 ± 2.3	5.2 ± 1.9	.32
Lateral mitral annular peak e′ velocity, cm/sec	7.3 ± 3.5	7.1 ± 2.5	.81

Data are expressed as mean ± SD.

Table 4 Periprocedural and 30-day mechanical and conduction outcomes of individuals with and without BSH

Variable	BSH+ (*n* = 55)	BSH− (*n* = 241)	*P*
Primary composite outcome*	22 (0.4)	116 (48.1)	.62
Modified primary composite outcome*	4 (7.3)	18 (7.5)	>.99
Valve pop-out	0 (0.0)	12 (5.0)	.23
Need for valve recapture	3 (5.5)	9 (3.7)	.44
Procedure aborted	0 (0.0)	1 (0.4)	>.99
Procedure converted to open surgery	0 (0.0)	0 (0.0)	>.99
Device embolization	1 (1.8)	1 (0.4)	.31
Postimplantation transaortic mean gradient, mm Hg	9.1 ± 4.6	10.3 ± 5.8	.21
Contrast volume, mL	149.9 ± 327.7	133.4 ± 103.7	.73
Need for pacemaker within 30-days	9 (16.4)	41 (17.0)	.41
Grade of paravalvular leak	1 (0–1)	1 (0–1)	.86
Postimplantation arrhythmia by type			
Atrial fibrillation	4 (7.3)	11 (4.6)	.91
Atrial flutter	0 (0.0)	0 (0.0)	>.99
Ventricular tachycardia	0 (0.0)	3 (1.2)	.57
Accelerated idioventricular rhythm	0 (0.0)	1 (0.4)	.83
Ectopic atrial rhythm	0 (0.0)	1 (0.4)	.83
New conduction disturbance by type	20 (36.4)	108 (44.8)	.55
Left bundle branch block	14 (25.5)	59 (24.5)	.76
Complete heart block	8 (14.5)	33 (13.7)	.75
First-degree atrioventricular block	0 (0.0)	6 (2.5)	.32
Second-degree atrioventricular block	0 (0.0)	2 (0.8)	>.99
Left anterior fascicular block	0 (0.0)	1 (0.4)	.83
Intraventricular conduction delay	0 (0.0)	3 (1.2)	.57
Right bundle branch block	0 (0.0)	2 (0.8)	.39
Asystole	1 (1.8)	1 (0.4)	.97

Data are expressed as mean ± SD.

*Includes the composite of valve pop-out, recapture, aborted procedure, conversion to an open procedure, device embolization, new conduction system disturbance, or need for a pacemaker within 30 days of the procedure. The modified primary composite outcome includes the above conditions, excluding new conduction system disturbance or need for a pacemaker within 30 days.

paravalvular leak between groups (BSH+ vs BSH−: median, 1 [interquartile range, 0–1] vs 1 [interquartile range, 0–1]; $P = .86$).

Adjusted Outcomes

After multivariable adjustment, BSH was not associated with the primary composite outcome (adjusted odds ratio BSH+ vs BSH−, 0.94; 95% CI, 0.42–2.11; $P = .88$.) After removing conduction system disturbance or need for PPM within 30 days from the composite outcome, BSH remained a nonsignificant predictor (adjusted odds ratio BSH+ vs BSH−, 1.49; 95% CI, 0.37–6.06; $P = .59$). Because image quality could influence the observed findings, we subsequently included image quality as a confounding variable in the multivariate models, without a change in the magnitude or direction of effect on the overall composite outcome (adjusted odds ratio BSH+ vs BSH−, 0.89; 95% CI, 0.40–2.02; $P = .78$).

DISCUSSION

Although adjacent to the left ventricular outflow tract and therefore theoretically important in the development of mechanical and conduction problems, the presence of BSH on preprocedural echocardiography was not associated with worsened periprocedural mechanical or 30-day conduction outcomes after TAVR in patients without preexisting pacemakers. BSH was associated with more frequent postdilation but similar postimplantation mean gradients.

Limited data currently exist on the outcomes of patients with BSH, limited in part by varying definitions of BSH.[11,19-23] Diaz *et al.*[11] evaluated the outcomes of patients with BSH in the Framingham Heart Study cohort over a 15-year follow-up period and after adjusting for cardiovascular risk factors and found that BSH was not independently associated with incident cardiovascular disease or mortality ($P > .30$ for both). Although BSH occurred predominantly in older individuals with higher systolic blood pressures, consistent with notion that BSH may represent a variant phenotypic response to long-standing hypertension,[15] it was not associated with worsened outcomes independent of hypertension in this community cohort. In the present cohort, a large majority (82.4%) had preexisting hypertension, with similar rates among those with and without BSH, and all patients had severe aortic stenosis. Although the occurrence of BSH in this sample was independent of hypertension or aortic stenosis severity (at least as measured by transvalvular gradients and the continuity equation), lack of information on the duration of both conditions and severity of hypertension could confound this finding. Of note, however, posterior wall thickness was not different between those with and those without BSH, suggesting that the development of BSH may be independent of overall hemodynamic effects on left ventricular hypertrophy.

To our knowledge, our study is the first to examine the relationship of BSH to mechanical and conduction outcomes after TAVR. A previous analysis of the Placement of Aortic Transcatheter Valves (PARTNER) trial and registry identified prosthesis size to left ventricular outflow tract gradient and prosthesis size to left ventricular diastolic diameter as predictors of pacemaker implantation,[24] but the relationship of BSH to outcomes after TAVR was previously unclear. In the present analysis, in patients without preexisting PPMs, the presence of BSH was unrelated to valve pop-out, valve recapture, requirement to abort the procedure or convert to an open surgery, device embolization, conduction disturbance or arrhythmia, a higher grade of paravalvular leak, postimplantation mean gradient, new conduction disturbance, or need for PPM within 30 days. Although BSH theoretically predisposes to these adverse outcomes because of narrowing of the left ventricular outflow tract and proximity to the conduction system,[11,14,15] as the bundle of His lies between the membranous septum and muscular basal septum, no such association was identified using the Framingham Heart Study definition of BSH. As modern TAVR implantation is often high, avoiding the muscular basal septum, it is possible that this procedural trend is responsible for the lack of the observed association between BSH and procedural or conduction outcomes across the range of implantation depths.

In this study, 50 TAVR patients (16.9%) ultimately received pacemakers within 30 days of the procedure. Although 8.8% of TAVR recipients in the PARTNER trial and registry ultimately received a PPM within 30 days, the predominant device used in this trial was the balloon-expandable ESV.[25] In the present study, 75% of valves implanted were the self-expandable MCV, which has been associated with average pacemaker implantation rates in meta-analyses of up to 25.8%.[26-28] Additionally, new conduction disturbance was demonstrated in 43.2% of individuals, of which 57.0% represented development of new left bundle branch block (overall rate in cohort of 24.7%). Although this rate is higher than the 10.5% noted in the PARTNER cohort, this may reflect predominant use of the MCV, which has been associated with rates of new left bundle branch block in the range of 35% to 65%.[29-34]

BSH was additionally associated with a number of baseline clinical and echocardiographic characteristics. First, those with BSH had lower measured creatinine. As information on estimated glomerular filtration rate is not available, it is possible that this represents sarcopenia in the BSH population, resulting in lower creatinine. On the other hand, it is possible that renal dysfunction is somehow related to the predisposition to develop symmetric hypertrophy rather than bulky upper septal hypertrophy. As selection bias regarding which patients with BSH undergo TAVR could potentially bias this association, it should be confirmed in other samples. On echocardiography, transmitral E-wave velocities were higher in those with BSH. This may reflect predominant early diastolic filling due to increased operating stiffness in those patients with BSH but should also be confirmed.

We also found that patients with BSH are more likely to require postdilation after TAVR placement. BSH has previously been associated with increased left ventricular outflow tract and annular ellipticity in aortic stenosis.[35] As the annular area is smaller for a given perimeter measurement in elliptical annuli,[36,37] oversizing a transcatheter heart valve on the basis of annular area may result in less oversizing in an elliptical versus circular annulus.[38] Both a lower percentage oversizing and an elliptical annulus have previously been associated with paravalvular leak,[39-41] a major reason for performing balloon postdilation after TAVR.[42,43] Thus, the higher rate of postdilation in patients with BSH may reflect attempts to correct malapposition of the transcatheter stent. Other reasons for postdilation could include operator preference, valvular calcification, or TAVR brand. Although the self-expanding MCV may require postdilation to correct malapposition more frequently and in subgroup analysis, this relationship was not observed in those receiving ESV devices ($P = .80$), the small number of those with ESVs and BSH ($n = 17$) limits conclusions about device subtypes. Conversely, this finding could be due to multiple hypothesis testing and should be confirmed in an independent sample. Postdilation in this setting has recently been shown to be safe,[38] and our study adds to this literature by demonstrating that postdilating a transcatheter valve in the setting of BSH is not clearly related to adverse outcomes from injury to the conduction system or immediate procedural concerns.

There were a number of limitations of this study worth considering. First, as this was a single-center evaluation at an experienced TAVR center, results may not generalize to other facilities or care settings. Second, as ventricular pacing may make identification of new complete heart block or conduction delay difficult, patients with preexisting PPMs were excluded from the analysis. Thus, the observed results may not generalize to this population. Third, as the study was retrospective, there may be residual confounding from unmeasured variables. Fourth, only 78.3% of the 438 patients had interpretable and nonmissing transthoracic echocardiographic images, which is similar to the 85% rate of interpretable images in our laboratory at BIDMC. Although these results are consistent with the quality of echocardiographic images seen in practice, and

adjusting for image quality in sensitivity analyses did not alter results, future studies should consider incorporating cardiac magnetic resonance imaging or computed tomographic measurements to avoid this issue. Fifth, as there is no clear consensus on how to define BSH, the results observed are highly dependent on the method of categorizing the exposure variable. Although the definition of BSH was congruent with that used in the Framingham Heart Study, it is possible that a different definition may result in discrepant conclusions, and this suggests the need to standardize a definition of BSH for future research. Sixth, given limited sample size and follow-up time, it is possible that a larger sample with longer follow-up may identify a relationship between BSH and outcomes. In particular, because of the small number of balloon-expandable, Lotus, and Direct Flow Medical valves included, it is possible that BSH may affect outcomes in patients receiving these valve types.

CONCLUSION

In this convenience sample of TAVR recipients at a large academic medical center, there was no effect of BSH on preprocedural echocardiography on periprocedural or 30-day adverse mechanical or conduction outcomes in patients without preexisting pacemakers. Those with BSH were more likely to receive postdilation. Standardization of definitions for BSH are needed to guide further research.

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